Salmonellosis and ulcerative colitis

A causal relationship or just a coincidence

Ali O. Karaoglu, MD, Vahit Yukselen, MD, Gunay T. Ertem, MD, Muhan Erkus, MD.

ABSTRACT

Coincidence of salmonellosis and ulcerative colitis is a rare clinical problem. Salmonella infection was reported to complicate the ulcerative colitis, as either facilitating its occurrence or activation. In this article, we present a case with salmonellosis whose clinicopathological findings also suggested ulcerative colitis. The patient improved rapidly after taking additional mesalazine to norfloxacin treatment. We conclude that salmonella infection might have either been coincidentally present or might have triggered an early ulcerative colitis in this patient who did not have history of inflammatory bowel diseases. In case of persistent severe diarrhea despite appropriate treatment, the possibility of a coincident inflammatory bowel disease such as ulcerative colitis should always be considered, especially in endemic regions for salmonellosis.


The etiopathogenesis of ulcerative colitis (UC) is not well defined. Food allergy, psychosomatic problems and immunological reactions against various bacteria antigens were reported in-between the probable etiological factors. Although various infectious agents were proposed in previous studies, none of them could be isolated. Some infectious agents including Salmonella species were reported to induce disease recurrence in UC. The relationship between salmonellosis and UC was emphasized in various case reports. We report a case with both salmonellosis and UC in whom salmonellosis might have triggered UC.

Case Report. A 40-year-old man was referred with fever, abdominal pain and bloody diarrhea of 30-40 times daily in the last 2-weeks. In another center, the patient’s stool cultures were negative, however, agglutinins of typhi O antigen was found positive at 1/320 dilutions and typhi H antigen was found positive at 1/80 dilutions. Treatment with ciprofloxacin 500mg bid for 5 days was administered but no improvement was observed. On admission, an oral temperature of 38.5°C was noted, and classic physical findings of enteric fever, such as pulse-temperature discordance, rose spots, headache, and splenomegaly were absent. Laboratory findings were erythrocyte sedimentation rate 43 mm/hour, white blood count was 7200/ml, hemoglobin 11 g/dl, agglutinins of typhi O and H antigens were both positive at 1/160, but the other titers were negative. Stool studies revealed profuse leukocytes and erythrocytes but no parasites. Rectoscopy revealed fragile, edematous, ground-like mucosa with petechia in some regions and bleeding upon slight contact. Rectum was
involved until the twentieth cm. Stool cultures were repeated thrice, and *Salmonella typhi* (*S*. *typhi*) was isolated in all on the second day. Therefore, norfloxacin 400mg once daily was started. Histopathological examination of rectal mucosa biopsies revealed diffuse lymphocytic infiltration in lamina propria, ulcers in mucosal surface, diminished mucus in the glands and dense neutrophilic and monocytic leucocyte infiltrations in crypt lumens. Considering these findings, a possible diagnosis of an accompanying UC could not be excluded (Figure 1). Mesalazine 2 g/day was added to norfloxacin treatment after which the patient had improved rapidly.

In the follow-up examinations at the fourth week, the patient was asymptomatic with negative stool cultures. Barium enema revealed severe mucosal granularity, spindle–like lesions considering ulceration and filling defects, narrowing at the transverse colon, splenic flexure and descending colon, but the other sites were intact (Figure 2). Mesalazine was reduced to 1 g/day maintenance dosage and the patient did not experience an activation of UC in the clinical follow-up.

**Discussion.** The presented case of salmonellosis had not improved under antibiotic treatment. Due to failure of previous treatment and rectoscopic and histological findings suggesting the possibility of an accompanying inflammatory bowel disease such as UC, mesalazine was started, which resulted with rapid remission. The rectoscopic appearance of *Salmonella colitis* can vary from relatively mild mucosal inflammation with hyperemia and contact bleeding to eroded surface and typical ulcers of elliptical shape with raised margins and punched-out appearance. These appearances are indistinguishable from UC. Some histological findings in *Salmonella colitis* such as superficial changes including necrosis and mixed inflammatory infiltrates are not specific for the causative agent. However, other histological appearances may be quite helpful in differentiating infections from the chronic inflammatory colitis. The presence of a chronic inflammatory infiltrates, architectural disturbances, and basal lymphoid aggregates are suggestive of UC. The loss of haustrations and the presence of diffuse small ulcers or erosions is radiological findings of *Salmonella colitis*, as of UC. But rectal sparing is considered as a characteristic feature of *Salmonella colitis*. In some previous articles, Salmonella infection was reported to complicate the UC, as either facilitating its occurrence or activation. In the presented case, we suggest that salmonellosis infection might have triggered an early UC. The absence of previous history for UC, clinicopathological findings suggesting UC isolation of *S*. *typhi* from the stool cultures and improvement of the patient’s condition after the addition of mesalazine treatment are supporting this possibility.

As a result, we conclude that salmonella infection might have been either coincidentally present or might have triggered UC in this patient who did not have a past inflammatory bowel disease history. This experience suggests that if severe diarrhea persists in salmonellosis despite appropriate treatment, the possibility of a coincident inflammatory bowel disease such as UC should always be considered, especially in endemic regions for salmonellosis.
References
